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* * * * * Welcome to STN International * * * * *

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NEWS 7 OCT 17 STN(R) AnaVist(TM), Version 1.01, allows the export/download
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* * * * * STN Columbus * * * * *

FILE 'HOME' ENTERED AT 11:27:30 ON 05 DEC 2005

=> FIL STNGUIDE

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

ENTRY

SESSION

FULL ESTIMATED COST

0.21

0.21

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FILE CONTAINS CURRENT INFORMATION.

LAST RELOADED: Dec 2, 2005 (20051202/UP).

=> FIL HOME

COST IN U.S. DOLLARS

SINCE FILE

ENTRY

TOTAL

SESSION

FULL ESTIMATED COST

0.06

0.27

FILE 'HOME' ENTERED AT 11:27:41 ON 05 DEC 2005

=> file biosis

COST IN U.S. DOLLARS

SINCE FILE

ENTRY

TOTAL

SESSION

FULL ESTIMATED COST

0.21

0.48

FILE 'BIOSIS' ENTERED AT 11:27:49 ON 05 DEC 2005

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FILE COVERS 1969 TO DATE.

CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT
FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 1 December 2005 (20051201/ED)

=> s (rem and sleep)

7284 REM

414 REMS

7515 REM

(REM OR REMS)

59961 SLEEP

91 SLEEPS

59985 SLEEP

(SLEEP OR SLEEPS)

L1 6890 (REM AND SLEEP)

=> s l1 and (blood or serum or sera)

2541806 BLOOD

598 BLOODS

2541882 BLOOD

(BLOOD OR BLOODS)

571469 SERUM

627 SERUMS

84086 SERA

12 SERAS

617792 SERUM

(SERUM OR SERUMS OR SERA OR SERAS)

84086 SERA

12 SERAS

84091 SERA

(SERA OR SERAS)

L2 613 L1 AND (BLOOD OR SERUM OR SERA)

=> s (diagnos? or prognos? or determin?) (3w) l1

PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH

FIELD CODE - 'AND' OPERATOR ASSUMED 'ETERMIN?) (3W) L1'

1141805 DIAGNOS?

160575 PROGNOS?

1524763 DETERMIN?

L3 1806 (DIAGNOS? OR PROGNOS? OR DETERMIN?) (3W) L1

=> s l3 and (blood or serum or sera)

2541806 BLOOD

598 BLOODS

2541882 BLOOD

(BLOOD OR BLOODS)

571469 SERUM

627 SERUMS

84086 SERA

12 SERAS

617792 SERUM

(SERUM OR SERUMS OR SERA OR SERAS)

84086 SERA

12 SERAS

84091 SERA

(SERA OR SERAS)

L4 183 L3 AND (BLOOD OR SERUM OR SERA)

=> s 14 and protein

1515860 PROTEIN

583567 PROTEINS

1743160 PROTEIN

(PROTEIN OR PROTEINS)

L5 4 L4 AND PROTEIN

=> d 15 1-4 kwic

L5 ANSWER 1 OF 4 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN

TI Inflammatory markers and **sleep** disturbance in major depression.

AB Objective: This study was conducted to **determine** whether immune activation Occurs in major depression, and to evaluate the associations between disordered **sleep** and markers of inflammation in patients with major depressive disorder. Methods: All-night polysomnography was obtained in patients with acute **Diagnostic** and Statistical Manual of Mental Disorders, 4th edition major depressive disorder (n = 22) and age-, gender-, and body weight-matched comparison controls (n = 18). After the onset of **sleep**, nocturnal **serum** levels of interleukin-6 (IL-6), soluble intercellular adhesion molecule (sICAM), monocyte chemotactic **protein** (MCP-1), and IL-6 Soluble receptor (IL-6sR) were sampled. Results: As compared with matched controls, depressed patients showed significant (p < .05) nocturnal elevations of circulating levels of IL-6 and sICAM. Both **sleep** latency and rapid eye movement (**REM**) density had moderate correlations with IL-6 and sICAM (r's gtoreq 0.30). Backward regression analyses indicated that **sleep** latency (beta = 0.34, p < .05) and **REM** density (beta = 0.27 p = .09) were better predictors of IL-6 than depressive Status. Similarly, **sleep** latency (beta = 0.27, p = .06) and **REM** density (beta = 0.32, p = .02) were also better predictors of sICAM. Conclusion: These findings support the hypothesis that **sleep** disturbance is associated with elevated levels of the inflammatory markers IL-6 and sICAM. This relationship was not accounted for by. . . findings suggest that the elevations in inflammatory markers found in depressive Subjects may be partially the result of disturbances of **sleep** initiation found in this Population.

IT .
Medicine, Medical Sciences); Neurology (Human Medicine, Medical Sciences); Psychiatry (Human Medicine, Medical Sciences)

IT Parts, Structures, & Systems of Organisms

serum: **blood** and lymphatics

IT Diseases

major depression: behavioral and mental disorders, pathology
Depression (MeSH)

IT Diseases

sleep disturbance: behavioral and mental disorders, nervous system disease, symptom
Sleep Disorders (MeSH)

IT Chemicals & Biochemicals

IL-6 soluble receptor [IL-6sR]; inflammatory markers; interleukin-6 [IL-6]; monocyte chemotactic **protein**-1 [MCP-1]; soluble intercellular adhesion molecule [sICAM]

IT Methods & Equipment

Diagnostic and Statistical Manual of Mental Disorders: clinical techniques, **diagnostic** techniques; polysomnography: clinical techniques, **diagnostic** techniques

IT Miscellaneous Descriptors

rapid eye movement

L5 ANSWER 2 OF 4 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN

AB. . . the other is hematopoietic PGDS (H-PGDS) in mast cells and Th2 lymphocytes. L-PGDS is the same as beta-trace, a major **protein** in human cerebrospinal fluid, and is also secreted into the seminal plasma

and plasma. The L-PGDS concentration in various body. . . and coronary atherosclerosis. H-PGDS is a cytosolic enzyme and is a member of the Sigma class of glutathione S-transferase. We **determined** the X-ray crystallographic structures of H-PGDS and L-PGDS. We also generated the gene-knockout (KO) mice and the human enzyme-overexpressing transgenic mice for each PGDS. L-PGDS-KO mice lacked PGE2-induced tactile allodynia and rebound of non-rapid eye movement **sleep** after **sleep** deprivation. Human L-PGDS-overexpressing transgenic mice showed an increase in non-rapid eye movement **sleep** due to accumulation of PGD2 in the brain after tail clipping. H-PGDS-KO mice showed an allergic reaction weaker than that. . .

IT Major Concepts

Behavior; **Blood** and Lymphatics (Transport and Circulation); Cardiovascular System (Transport and Circulation); Cell Biology; Endocrine System (Chemical Coordination and Homeostasis); Enzymology (Biochemistry. . . (Neural Coordination); Reproductive System (Reproduction); Urinary System (Chemical Coordination and Homeostasis)

IT Parts, Structures, & Systems of Organisms

Th2 lymphocyte: **blood** and lymphatics, immune system; cerebrospinal fluid: nervous system; mast cell: immune system; plasma: **blood** and lymphatics; seminal plasma: reproductive system

IT Diseases

allergy: immune system disease, etiology
Hypersensitivity (MeSH)

IT Diseases

coronary atherosclerosis: heart disease,. . .

IT Methods & Equipment

x-ray crystallography: crystallographic techniques, laboratory techniques

IT Miscellaneous Descriptors

hematopoiesis; non-**REM sleep**

L5 ANSWER 3 OF 4 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN

TI Inhibition of tumor necrosis factor in the brain suppresses rabbit **sleep**.

AB Tumor necrosis factor (TNF) is a cytokine that possesses many biological activities, including enhancement of non-rapid-eye-movement **sleep** (NREMS). The role of endogenous TNF in the regulation of spontaneous **sleep** is unknown. If TNF is involved in **sleep** regulation, then reduction of endogenous TNF should suppress spontaneous **sleep**. A soluble TNF-binding protein I (TNF-BP I) and a synthetic fragment of TNF-BP I, TNF-R-(159-178), that contains the biologically active region of TNF-BP I, were used. These substances bind TNF and possess TNF-inhibitory activity; their effects on rabbit **sleep** after intracerebroventricular injection were **determined** across a 6-h recording period. Two doses of TNF-BP I (0.05 mu-g and 0.5 mu-g) were administered; the higher dose. . . mu-g) were used. The 25 mu-g and 50 mu-g doses significantly suppressed NREMS. The highest dose (50 mu-g) also decreased **REM sleep**. These results are consistent with the hypothesis that endogenous brain TNF is involved in the regulation of normal **sleep**.

IT Major Concepts

Behavior; Biochemistry and Molecular Biophysics; **Blood** and Lymphatics (Transport and Circulation); Endocrine System (Chemical Coordination and Homeostasis); Nervous System (Neural Coordination); Sense Organs (Sensory Reception)

IT Miscellaneous Descriptors

RAPID EYE MOVEMENT **SLEEP**; TUMOR NECROSIS FACTOR-BINDING PROTEIN-1

L5 ANSWER 4 OF 4 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN

AB. . . a muscular habitus, and muscle stiffness and myokymia were found in all muscles of the extremities. Her stiffness persisted during **sleep**. Her calf muscles were large and a contracture was noticed in ankle joints. There was no evidence of wasting and. . . of the CPK linked IgA was lamda type. All other laboratory tests were normal for the following: urinalysis, ESR, a **blood** count, liver function, kidney function, glucose, rheumatoid factor, CRP, thyroid function, parathyroid function, **serum** electrolytes, ECG, EEG, cranial CT, without slight elevation of IgA, and CSF **protein**. In needle EMG

and surface EMG spontaneous discharges were recorded at rest. These discharges consist of normal motor unit potentials, . . . needle EMG, myotonic discharge was not observed. Nerve conduction velocities were within normal ranges. According to these data, she was **diagnosed** as having Issacs' syndrome (continuous muscle fiber activity syndrome). Carbamazepine, 200 mg daily was administered and showed a dramatic reversal. . . to carbamazepine. Polysomnography was examined before and after treatment. Before treatment, suppression of muscle fiber discharges was not recognized during **REM sleep**. After treatment, suppression of muscle fiber discharges appeared and % stage was elevated (% stage IV, 3.6% → 11.4%). It was suspected that continuous muscle fiber discharges throughout the **sleep** had an influence on the depth of the **sleep** stage. There have been reports of some 40 cases of syndrome of continuous muscle fiber activity. A number of studies. . .

=> d 15 1-3 ibib, iabs

L5 ANSWER 1 OF 4 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 2005:208458 BIOSIS
DOCUMENT NUMBER: PREV200500212330
TITLE: Inflammatory markers and **sleep** disturbance in major depression.
AUTHOR(S): Motivala, Sarosh J. [Reprint Author]; Sarfatti, Avishay; Olmos, Luis; Irwin, Michael R.
CORPORATE SOURCE: Inst Neuropsychiat Cousins Ctr Psychoneuroimmunol, Univ Calif Los Angeles, 300 Med Plaza, Suite 3160A, Los Angeles, CA, 90095, USA
smotivala@mednet.ucla.edu
SOURCE: Psychosomatic Medicine, (March 2005) Vol. 67, No. 2, pp. 187-194. print.
ISSN: 0033-3174 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 1 Jun 2005
Last Updated on STN: 1 Jun 2005

ABSTRACT:

Objective: This study was conducted to **determine** whether immune activation Occurs in major depression, and to evaluate the associations between disordered **sleep** and markers of inflammation in patients with major depressive disorder.

Methods: All-night polysomnography was obtained in patients with acute *****Diagnostic***** and Statistical Manual of Mental Disorders, 4th edition major depressive disorder (n = 22) and age-, gender-, and body weight-matched comparison controls (n = 18).

After the onset of **sleep**, nocturnal **serum** levels of interleukin-6 (IL-6), soluble intercellular adhesion molecule (sICAM), monocyte chemotactic **protein** (MCP-1), and IL-6 Soluble receptor (IL-6sR) were sampled.

Results: As compared with matched controls, depressed patients showed significant ($p < .05$) nocturnal elevations of circulating levels of IL-6 and sICAM.

Both **sleep** latency and rapid eye movement (**REM**) density had moderate correlations with IL-6 and sICAM (r 's $gtoreq$ 0.30).

Backward regression analyses indicated that **sleep** latency ($\beta = 0.34$, $p < .05$) and **REM** density ($\beta = 0.27$ $p = .09$) were better predictors of IL-6 than depressive Status.

Similarly, **sleep** latency ($\beta = 0.27$, $p = .06$) and **REM** density ($\beta = 0.32$, $p = .02$) were also better predictors of sICAM.

Conclusion: These findings support the hypothesis that **sleep** disturbance is associated with elevated levels of the inflammatory markers IL-6 and sICAM.

This relationship was not accounted for by other confounding factors such as age and body weight.

These findings suggest that the elevations in inflammatory markers found in depressive Subjects may be partially the result of disturbances of

*****sleep***** initiation found in this Population.

L5 ANSWER 2 OF 4 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN

ACCESSION NUMBER: 2004:140670 BIOSIS
DOCUMENT NUMBER: PREV200400135658
TITLE: Functional analyses of lipocalin-type and hematopoietic
prostaglandin D synthases.
AUTHOR(S): Urade, Yoshihiro [Reprint Author]; Eguchi, Naomi [Reprint
Author]; Aritake, Kosuke [Reprint Author]; Hayaishi, Osamu
[Reprint Author]
CORPORATE SOURCE: Department of Molecular Behavioral Biology, Osaka
Bioscience Institute, 6-2-4 Furuedai, Suita, Osaka,
565-0874, Japan
SOURCE: Folia Pharmacologica Japonica, (January 2004) Vol. 123, No.
1, pp. 5-13. print.
ISSN: 0015-5691 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: Japanese
ENTRY DATE: Entered STN: 10 Mar 2004
Last Updated on STN: 10 Mar 2004

ABSTRACT:

Prostaglandin (PG) D synthase (PGDS) catalyzes the isomerization of PGH2 to
PGD2, which acts as an endogenous somnogen and an allergic mediator.
There are two distinct types of PGDS: one is lipocalin-type PGDS (L-PGDS)
localized in the central nervous system, male genitals, and heart; and the
other is hematopoietic PGDS (H-PGDS) in mast cells and Th2 lymphocytes.
L-PGDS is the same as beta-trace, a major protein in human
cerebrospinal fluid, and is also secreted into the seminal plasma and plasma.
The L-PGDS concentration in various body fluids is useful as a marker for
various diseases such as renal failure and coronary atherosclerosis.
H-PGDS is a cytosolic enzyme and is a member of the Sigma class of glutathione
S-transferase.
We determined the X-ray crystallographic structures of H-PGDS and
L-PGDS.
We also generated the gene-knockout (KO) mice and the human
enzyme-overexpressing transgenic mice for each PGDS.
L-PGDS-KO mice lacked PGE2-induced tactile allodynia and rebound of non-rapid
eye movement sleep after sleep deprivation.
Human L-PGDS-overexpressing transgenic mice showed an increase in non-rapid eye
movement sleep due to accumulation of PGD2 in the brain after tail
clipping.
H-PGDS-KO mice showed an allergic reaction weaker than that of the wild-type
mice.

L5 ANSWER 3 OF 4 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 1996:80564 BIOSIS
DOCUMENT NUMBER: PREV199698652699
TITLE: Inhibition of tumor necrosis factor in the brain suppresses
rabbit sleep.
AUTHOR(S): Takahashi, Satoshi; Tooley, Dawn D.; Kapas, Levente; Fang,
Jidong; Seyer, Jerome M.; Krueger, James M. [Reprint
author]
CORPORATE SOURCE: Dep. Physiol. Biophysics, Univ. Tennessee, Memphis, TN
38163, USA
SOURCE: Pfluegers Archiv European Journal of Physiology, (1995)
Vol. 431, No. 2, pp. 155-160.
CODEN: PFLABK. ISSN: 0031-6768.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 27 Feb 1996
Last Updated on STN: 27 Feb 1996

ABSTRACT:

Tumor necrosis factor (TNF) is a cytokine that possesses many biological
activities, including enhancement of non-rapid-eye-movement sleep
(NREMS).
The role of endogenous TNF in the regulation of spontaneous sleep is
unknown.
If TNF is involved in sleep regulation, then reduction of endogenous
TNF should suppress spontaneous sleep.
A soluble TNF-binding protein I (TNF-BP I) and a synthetic fragment
of TNF-BP I, TNF-R-(159-178), that contains the biologically active region of
TNF-BP I, were used.
These substances bind TNF and possess TNF-inhibitory activity; their effects on

rabbit **sleep** after intracerebroventricular injection were
 determined across a 6-h recording period.
 Two doses of TNF-BP I (0.05 mu-g and 0.5 mu-g) were administered; the higher
 dose of TNF-BP I significantly decreased NREMS.
 Four doses of TNF-R-(159-178) (0.25 mu-g, 2.5 mu-g, 25 mu-g and 50 mu-g) were
 used.
 The 25 mu-g and 50 mu-g doses significantly suppressed NREMS.
 The highest dose (50 mu-g) also decreased **REM sleep**.
 These results are consistent with the hypothesis that endogenous brain TNF is
 involved in the regulation of normal **sleep**.

=> s l4 and electrophoresis
 201413 ELECTROPHORESIS
 L6 0 L4 AND ELECTROPHORESIS

=> s l4 and marker
 172141 MARKER
 145983 MARKERS
 280535 MARKER
 (MARKER OR MARKERS)
 L7 8 L4 AND MARKER

=> d l7 1-8 kwic

L7 ANSWER 1 OF 8 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
 TI Inflammatory **markers** and **sleep** disturbance in major
 depression.

AB Objective: This study was conducted to **determine** whether immune
 activation Occurs in major depression, and to evaluate the associations
 between disordered **sleep** and **markers** of inflammation
 in patients with major depressive disorder. Methods: All-night
 polysomnography was obtained in patients with acute **Diagnostic**
 and Statistical Manual of Mental Disorders, 4th edition major depressive
 disorder (n = 22) and age-, gender-, and body weight-matched comparison
 controls (n = 18). After the onset of **sleep**, nocturnal
serum levels of interleukin-6 (IL-6), soluble intercellular
 adhesion molecule (sICAM), monocyte chemotactic protein (MCP-1), and IL-6
 Soluble receptor (IL-6sR) were sampled.. . with matched controls,
 depressed patients showed significant (p < .05) nocturnal elevations of
 circulating levels of IL-6 and sICAM. Both **sleep** latency and
 rapid eye movement (**REM**) density had moderate correlations with
 IL-6 and sICAM (r's gtoreq 0.30). Backward regression analyses indicated
 that **sleep** latency (beta = 0.34, p < .05) and **REM**
 density (beta = 0.27 p = .09) were better predictors of IL-6 than
 depressive Status. Similarly, **sleep** latency (beta = 0.27, p =
 .06) and **REM** density (beta = 0.32, p = .02) were also better
 predictors of sICAM. Conclusion: These findings support the hypothesis
 that **sleep** disturbance is associated with elevated levels of the
 inflammatory **markers** IL-6 and sICAM. This relationship was not
 accounted for by other confounding factors such as age and body weight.
 These findings suggest that the elevations in inflammatory **markers**
 found in depressive Subjects may be partially the result of disturbances
 of **sleep** initiation found in this Population.

IT .
 Medicine, Medical Sciences); Neurology (Human Medicine, Medical
 Sciences); Psychiatry (Human Medicine, Medical Sciences)

IT Parts, Structures, & Systems of Organisms
serum: blood and lymphatics

IT Diseases
 major depression: behavioral and mental disorders, pathology
 Depression (MeSH)

IT Diseases
sleep disturbance: behavioral and mental disorders, nervous
 system disease, symptom
Sleep Disorders (MeSH)

IT Chemicals & Biochemicals
 IL-6 soluble receptor [IL-6sR]; inflammatory **markers**;
 interleukin-6 [IL-6]; monocyte chemotactic protein-1 [MCP-1]; soluble
 intercellular adhesion molecule [sICAM]

IT Methods & Equipment
Diagnostic and Statistical Manual of Mental Disorders:
clinical techniques, **diagnostic** techniques; polysomnography:
clinical techniques, **diagnostic** techniques

IT Miscellaneous Descriptors
rapid eye movement

L7 ANSWER 2 OF 8 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI The association between interleukin-6, **sleep**, and demographic
characteristics.

AB We examined the relationship between the pro-inflammatory cytokine IL-6
and **sleep** architecture in 10 healthy men and women.
Blood was drawn in the early morning for assessment of IL-6
followed by nocturnal **sleep** monitoring with polysomnography.
Sleep records were scored for **sleep** stages using
standard criteria. Morning IL-6 levels were positively correlated with
REM latency after **sleep** onset ($p = .31$, $p = .01$), percent
(%) stage I **sleep** ($p = .23$, $p = .053$) % wake after **sleep**
onset (WASO) ($p = .29$, $p < .05$). IL-6 levels were negatively correlated
with **sleep** efficiency ($p = -.36$, $p < .01$) and slow wave
sleep (SWS) ($p = -.26$, $p < .05$). After controlling for demographic
variables including race, gender, age, and BMI multiple hierarchical
regression analyses revealed that morning IL-6 levels accounted for a
significant portion of the variance of **REM** latency ($p < .01$)
sleep efficiency ($p < .01$), and % WASO ($p = .01$). IL-6 was no
longer associated with % stage I **sleep**, SWS, and total
sleep time after controlling for the demographic characteristics.
These findings suggest that the inflammatory marker IL-6 is
associated with **sleep** quality and that certain individual
characteristics such as race, gender, and age modify that relationship.
Higher IL-6 levels were associated with lower quality of **sleep**
among healthy asymptomatic men and women. Copyright 2004 Elsevier Inc.
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IT . . .
Biochemistry and Molecular Biophysics; Clinical Immunology (Human
Medicine, Medical Sciences); Epidemiology (Population Studies)

IT Parts, Structures, & Systems of Organisms
blood: **blood** and lymphatics

IT Chemicals & Biochemicals
interleukin-6: pro-inflammatory cytokine

IT Methods & Equipment
polysomnography: clinical techniques, **diagnostic** techniques

IT Miscellaneous Descriptors
demographic characteristic; **sleep** pattern

L7 ANSWER 3 OF 8 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
AB . . . is also secreted into the seminal plasma and plasma. The L-PGDS
concentration in various body fluids is useful as a **marker** for
various diseases such as renal failure and coronary atherosclerosis.
H-PGDS is a cytosolic enzyme and is a member of the Sigma class of
glutathione S-transferase. We **determined** the X-ray
crystallographic structures of H-PGDS and L-PGDS. We also generated the
gene-knockout (KO) mice and the human enzyme-overexpressing transgenic
mice for each PGDS. L-PGDS-KO mice lacked PGE2-induced tactile allodynia
and rebound of non-rapid eye movement **sleep** after **sleep**
deprivation. Human L-PGDS-overexpressing transgenic mice showed an
increase in non-rapid eye movement **sleep** due to accumulation of
PGD2 in the brain after tail clipping. H-PGDS-KO mice showed an allergic
reaction weaker than that. . . .

IT Major Concepts
Behavior; **Blood** and Lymphatics (Transport and Circulation);
Cardiovascular System (Transport and Circulation); Cell Biology;
Endocrine System (Chemical Coordination and Homeostasis); Enzymology
(Biochemistry). . . (Neural Coordination); Reproductive System
(Reproduction); Urinary System (Chemical Coordination and Homeostasis)

IT Parts, Structures, & Systems of Organisms
Th2 lymphocyte: **blood** and lymphatics, immune system;
cerebrospinal fluid: nervous system; mast cell: immune system; plasma:
blood and lymphatics; seminal plasma: reproductive system

IT Diseases

allergy: immune system disease, etiology
Hypersensitivity (MeSH)

IT Diseases
coronary atherosclerosis: heart disease, . . .
failure: urologic disease
Kidney Failure (MeSH)

IT Chemicals & Biochemicals
glutathione S-transferase [EC 2.5.1.18]; lipocalin; prostaglandin D
synthase [EC 5.3.99.2]: disease **marker**, hematopoietic;
lipocalin-type; prostaglandin H-2

IT Methods & Equipment
x-ray crystallography: crystallographic techniques, laboratory
techniques

IT Miscellaneous Descriptors
hematopoiesis; non-REM **sleep**

L7 ANSWER 4 OF 8 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI Arterial stiffness increases during obstructive **sleep** apneas.
AB Study Objectives: Obstructive **sleep** apnea (OSA) appears to be an
independent risk factor for diurnal systemic hypertension, but the
specific biologic **markers** for this association have not been
well established. Increased arterial stiffness is an important measure of
increased left ventricular load. . . . However, arterial stiffness has
not been measured in association with obstructive apneas in patients with
OSA, nor related to systemic **blood** pressure (BP) activity in
this setting. Our objective was to test the hypothesis that arterial
stiffness may be utilized as. . . (2) such increased stiffness may
occur in the absence of acute BP increase. Design: Prospective,
cross-sectional. Setting: A tertiary-care university-based **sleep**
and ventilatory disorders center. Patients: Forty-four normo- and
hypertensive adult patients (11 women, 33 men) with polysomnographically
diagnosed moderate to severe OSA. Interventions: N/A.
Measurements and Results: Beat-to-beat BP was recorded from the radial
artery by applanation tonometry. . . first 15 cardiac cycles following
apnea termination ("post apnea"). Mean AAI (+-SD) for the group was
significantly increased during NREM **sleep** from early apnea to
late apnea (12.02+-2.70% vs 13.35+-3.54%, p<0.05, ANOVA). During
REM (analyzed in 20 patients), AAI again significantly increased
from early apnea to late apnea (11.75+-2.81% vs 13.43+-4.97%).
Conversely, neither mean. . . arterial BP was significantly changed
from early apnea to late apnea in NREM (SBP 130+-14 mmHg vs 129+-14 mmHg)
or REM (SBP 128+-22 mmHg vs 127+-21 mmHg). Conclusions:
Arterial stiffness increases acutely during obstructive apneas in both
NREM and REM **sleep**, in the absence of measurable BP
change. These data suggest that arterial stiffness may be a sensitive
measure of acute. . . .

IT . . .
Sciences)

IT Parts, Structures, & Systems of Organisms
artery: circulatory system

IT Diseases
hypertension: vascular disease
Hypertension (MeSH)

IT Diseases
obstructive **sleep** apnea: respiratory system disease,
complications
Sleep Apnea, Obstructive (MeSH)

IT Methods & Equipment
arterial augmentation index: clinical techniques, **diagnostic**
techniques; polysomnography: clinical techniques, **diagnostic**
techniques

IT Miscellaneous Descriptors
arterial stiffness: modulation; systolic BP [systolic **blood**
pressure]: modulation

L7 ANSWER 5 OF 8 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI Light-dark difference in arterial pressure variability during REM
sleep in the rat.
AB We have observed mean arterial pressure (MAP) variability during rapid eye
movement (REM) **sleep** and brain temperature (Tb) in the

rat during both light and dark periods over 24 h. MAP was measured using a telemetric device with a computer data capture and analysis system. As markers of MAP variability, the maximum and coefficient of variation (CV%) of MAP during REM sleep were determined. The following results were obtained: (a) there was a light-dark difference in MAP during non-REM (NREM) sleep and Tb during both NREM and REM sleep; (b) the increase of MAP in going from NREM to REM sleep in the light period was greater than that in the dark period, whereas the increase of Tb in the light period was not different from that in the dark period; (c) the maximum and CV% for MAP during REM sleep in the light period were greater than those in the dark period; (d) there was a negative correlation between the average Tb and MAP CV% during REM sleep. We suggest that phasic fluctuation of MAP during REM sleep may be influenced, in part, by a factor independent of sleep mechanisms. Key Words: Arterial pressure-Brain temperature-Rapid eye movement sleep -Rat-Circadian rhythm.

IT Major Concepts

Behavior; Biochemistry and Molecular Biophysics; Biosynchronization; Blood and Lymphatics (Transport and Circulation); Cardiovascular System (Transport and Circulation); Cell Biology; Metabolism; Nervous System (Neural Coordination); Physiology; Radiation Biology; . . .

IT Miscellaneous Descriptors

BRAIN TEMPERATURE; CHRONOBIOLOGY; RAPID EYE MOVEMENT SLEEP

L7 ANSWER 6 OF 8 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI Genetic influences on EEG sleep and the human circadian clock: A twin study.

AB The study of neuroendocrine and sleep abnormalities in major depressive disorders has been the focus of major interest in the past few years. However, while sleep and neuroendocrine research in neuropsychiatric disorders has progressed considerably during the last few years, conceptional and methodological advances in sleep and neuroendocrine physiology are still needed for further understanding of the basic aspects of sleep and to clarify the control and significance of the temporal fluctuations of the neuroendocrine systems. In particular, identification of the genetic mechanisms governing sleep regulation are of interest. In this respect, twin studies constitute a powerful method for identifying genetic influences on human physiological variables. In a first study, we explored the sleep patterns of 26 pairs of noncohabiting normal male twins (both mono- and dizygotic). The results indicate that a significant genetic effect is found for some sleep variables. Stages 2, 4, and delta sleep as well as waking are substantially determined by genetic factors, in contrast to stage REM which seems to be mainly affected by nongenetic influences. These data thus provide consistent evidence that some aspects of human sleep are genetically determined. In a second study we analyzed the 24-hour profile of plasma cortisol in 21 pairs of male twins. The 24-hour profile of plasma cortisol is the most widely used marker of the human circadian clock: Its study offers the possibility of assessing the status of the human circadian clock and of determining whether genetic factors affect human circadian rhythmicity. In the protocol, blood was sampled every 15 min and circadian rhythmicity was characterized by measures of amplitude, phase, and overall waveshape. A genetic. . .

IT Major Concepts

Behavior; Biosynchronization; Blood and Lymphatics (Transport and Circulation); Clinical Chemistry (Allied Medical Sciences); Endocrine System (Chemical Coordination and Homeostasis); Genetics; Metabolism; Nervous System. . .

IT Miscellaneous Descriptors

CORTISOL; DELTA SLEEP; ELECTROENCEPHALOGRAPHY; NOCTURNAL NADIR; PLASMA LEVEL; PULSATILE TEMPORAL VARIABILITY; RAPID EYE MOVEMENT; REGULATION; STAGE 2; STAGE 4; WAKING

L7 ANSWER 7 OF 8 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI USEFULNESS OF SLEEP AND NEUROENDOCRINE TESTS AS BIOLOGICAL

MARKERS OF DEPRESSION IN CHILDREN AND ADOLESCENTS.

AB In this article, we systematically reviewed the results of application of biological **markers** of depression to children and adolescents. Concerning **sleep** EEG, only three studies on a total of twelve among 267 depressed children and adolescents aged 6 to 19 years found the typical **sleep** abnormalities described in depressed adults (eg, shortened **REM** latency and decreased **sleep** efficiency). Most authors insisted on the age-related **sleep** changes as a major confounding factor. Two studies of the effect of antidepressant therapy on **sleep** showed a decrease in **sleep** efficiency but a discrepancy in the evolution of **REM** latency. Concerning the dexamethasone suppression test, twenty studies including 374 depressed children and adolescents (3-20 years) and 533 psychiatric controls. . . be considered as interesting, despite the lack of agreement among authors on various methodological parameters (dose of dexamethasone, times of **blood** sampling, method of cortisol assay...) and the composition of control groups which often comprise subjects presenting disorders very close to. . . in two studies, showed limited interest. In contrast, the study of growth hormone secretion, performed in one centre, could present **diagnostical** usefulness. In conclusion, biological **markers** of depression in children and adolescents should still be considered as research tools and be part of a multidisciplinary approach.

L7 ANSWER 8 OF 8 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI RED **BLOOD** CELL-PLASMA CHOLINE RATIO IN ELDERLY DEPRESSED AND
DEMENTED PATIENTS.

AB In further study of red **blood** cell (RBC) and plasma choline concentrations in 160 elderly subjects, we found no significant differences in RBC/plasma choline ratios among. . . RBC/plasma choline ratios > 1.9. Thus, it now appears that static RBC choline levels cannot be recommended as a specific **marker** of Alzheimer's dementia. However, within subgroups of these **diagnostic** categories, **determined** by RBC/plasma choline ratios ≤ 1.9 or > 1.9 , consistent differences in electroencephalographic (EEG) **sleep** measures were found. The subgroup of demented patients with a RBC/plasma choline ratio > 1.9 was more impaired on the Blessed Dementia Rating Scale and had less rapid eye movement (**REM**) **sleep** than the subgroup with a choline ratio ≤ 1.9 . Similarly, depressives with a choline ratio ≤ 1.9 had a lower **REM** latency than depressives with a choline ratio > 1.9. Finally, depressed-demented (i.e., mixed-symptom) patients with a choline ratio > 1.9 showed less **sleep** continuity disturbance but more indeterminate non-**REM sleep** (reflecting loss of spindles and K-complexes) than those with lower choline ratios. These differences parallel those previously reported for **diagnostically** 'pure' depressed and demented patients, and they suggest a possible link between peripheral RBC/plasma choline measures and central nervous system function as reflected in **sleep** physiological alterations.

IT Major Concepts
Behavior; **Blood** and Lymphatics (Transport and Circulation);
Geriatrics (Human Medicine, Medical Sciences); Metabolism; Neurology
(Human Medicine, Medical Sciences); Pathology; Psychiatry (Human
Medicine, . . .
IT Miscellaneous Descriptors
RAPID EYE MOVEMENT **SLEEP**

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FILE 'HOME' ENTERED AT 11:27:41 ON 05 DEC 2005

FILE 'BIOSIS' ENTERED AT 11:27:49 ON 05 DEC 2005

L1 6890 S (REM AND SLEEP)
L2 613 S L1 AND (BLOOD OR SERUM OR SERA)
L3 1806 S (DIAGNOS? OR PROGNOS? OR DETERMIN?) (3W) L1

L4 183 S L3 AND (BLOOD OR SERUM OR SERA)
L5 4 S L4 AND PROTEIN
L6 0 S L4 AND ELECTROPHORESIS
L7 8 S L4 AND MARKER

=> s l4 and (proteinase)
27239 PROTEINASE
9715 PROTEINASES
31551 PROTEINASE
(PROTEINASE OR PROTEINASES)
L8 0 L4 AND (PROTEINASE)

=> s l4 and (glycosylated)
18637 GLYCOSYLATED
L9 0 L4 AND (GLYCOSYLATED)

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COST IN U.S. DOLLARS	ENTRY	SESSION
FULL ESTIMATED COST	60.65	61.13

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